

Modern Journal of Social Sciences and Humanities
ISSN: 2795-4846 Vol. 5 (2022)
https://mjssh.academicjournal.io/index.php/mjssh

INSULIN-LIKE GROWTH FACTOR AND HYPERTENSION

Soliyev Alisher Urokovich
Assistant, Bukhara State Medical Institute Department Of Internal Medicine and Endocrinology

Annatation: Insulin-like growth factor-1 (IGF-1) polypeptide is a representative of growth factors and is close to insulin by its physiological effects. This factor plays an important role in the mechanisms of regulation of the structure and function of the myocardium and blood vessels. IGF-1 is detected in the processes of hypertrophy of the heart and blood vessels. A number of authors link IGF-1 to prognostic biological signs of the development of heart failure.

To date, the pathogenetic role of IGF-1 in the development of injuries of the cardiovascular system, including arterial hypertension, type 2 diabetes and their combination, remains unclear. In this regard, it is of great scientific interest to determine the relationship between the level of IGF-1 in the blood and the structural and functional parameters of the heart and carotid artery in patients with type 2 diabetes, hypertension. Treatment with metformin in patients with hypertension with type 2 diabetes resulted in a significant decrease in IGF-1 levels in the blood. This was particularly evident in patients with concentric left ventricular hypertrophy prior to treatment and with elevated IGF-1 levels. However, the addition of long-acting glyclazide to metformin, no decrease in IGF-1 levels in the blood of patients after treatment was not observed [1].

Keywords: diabetes mellitus, prevention, diagnosis, treatment.

Both hypertension and obesity lead to structural and functional remodeling of the myocardium and the formation of left ventricular hypertrophy associated with the development of interstitial fibrosis. Cardiovascular fibrosis is a humoral process in which angiotensin II (ATII), endothelin-I, and aldosterone play a central role.

Angiotensin II stimulates type I collagen production and stimulates the involvement of profibrogenic peptide growth factors such as insulin-like growth factor-1 (IGF-1) and transformational growth factor b1, which alter ATII response. Activation of these humoral factors leads to the proliferation of fibroblasts and the development of imbalances in the process of collagen synthesis and degradation, its excessive accumulation in the interstitial space. An important representative of profibrogenic growth factors is IGF-1, which is produced in the liver, cardiomyocytes, smooth muscle cells, and fibroblasts under the influence of growth hormone. IGF-1 is believed to be partially structurally close to insulin, has an insulin-like metabolic effect, lowers glucose levels, and reduces insulin resistance. This factor plays an important role in the mechanisms that regulate the structure and function of the myocardium and blood vessels. Experiments have shown that IGF-1 plays a leading role in the protection of cardiomyocytes from apoptosis, both in vivo and in vitro. In experimental myocardial infarction in mice, a decrease in apoptosis was observed against the background of increased production of IGF -1, and in the absence of IGF-1 decreased DNA synthesis and increased apoptosis in cardiomyocytes. In recent years, the association of IGF-1 as an independent risk factor in cardiovascular disease has been discussed, but the results of these studies are highly controversial. A number of studies have shown that increased IGF -1 levels lead to a higher risk of developing ischemic heart disease, while other studies have shown that lower IGF-1 levels increase the risk of developing ischemic heart disease and death. There are studies showing the role of IGF-1 as a biological marker of prognostic significance

for the development of heart failure. The results of clinical studies evaluating the level of IGF-1 in the blood of patients with hypertension are also not the same.

Thus, the study found that IGF-1 concentrations were higher in patients with hypertension than in normotonics, and in other studies, in contrast, in patients with hypertension, compared with controls, decreased, or with type 2 diabetes and arterial hypertension. in patients with pathological types of myocardial remodeling, its low level was noted. However, studies aimed at evaluating the effects of profibrotic growth factors on hemodynamic parameters and left ventricular remodeling in patients with arterial hypertension and metabolic syndrome are rare. Among the humoral factors that stimulate the growth of cardiomyocytes and myocardial fibroblasts, the sympathetic and renin-angiotensin systems play an important role, and the contribution of insulin and insulin-like growth factors in the regulation of connective tissue production has been studied. , especially in metabolic syndrome. IGF-1 is the main representative of the family of insulin-like growth factors, carries out endocrine, autocrine and paracrine regulation of growth processes and is actively involved in anabolic reactions in connective tissue, muscle and heart. IGF-1 levels are regulated by growth hormone, and its concentration depends on the effects of both growth hormone and sex steroids, thyroid hormones, glucocorticoids, and insulin on the liver. At the same time, insulin and estrogens increase the synthesis of IGF-1 in the liver, while glucocorticoids reduce it, which provides a synergistic effect of insulin, somatotropin, sex and thyroid hormones on the growth and differentiation of cells and body tissues. IGF-1 is known as insulin-like growth factor because of its ability to stimulate glucose absorption by muscle and adipose tissue in a very similar and insulin-like manner to proinsulin [28,29,30]. The role of IGF-1 factor in the development of many pathological processes in vascular disease has been demonstrated in experimental and clinical studies [18, 19, 30]. However, the effect of IGF-1 on hemodynamics and the prognosis of cardiovascular disease are highly contradictory. Thus, low levels of IGF-1 are associated with a risk of coronary heart disease (CHD), including cardiovascular death. However, there are conflicting data, suggesting that individuals with high levels of IGF-1 are more likely to develop UIC disease [8]. On the other hand, in a study evaluating the relationship between life expectancy and IGF-1 levels in older men, IGF-1 levels were higher in both the longest-lived general population and those with a poor cardiovascular history. which was. The literature data showing changes in IGF-1 levels in patients with hypertension are also contradictory. In addition to studies showing an increase in IGF-1 activity in hypertensive patients compared to normotonic patients, there are also studies showing a decrease in IGF-1 concentration in the blood of hypertensive patients compared to control groups. Changes in IGF-1 levels in the blood of patients with AG and MS were found to be related to the severity of obesity and to reflect myocardial left ventricular remodeling characteristics. Level ventricular concentric hypertrophy in grade 1 obesity is characterized by hyperproduction of IGF-1.

and eccentric left ventricular hypertrophy at levels 2-3 of obesity is apparently associated with decreased IGF-1 synthesis, the lack of which contributes to increased cardiomyocyte apoptosis and the development of myocardial fibrosis. The results are consistent with data from other studies in which a decrease in IGF-1 activity was noted in AH patients with severe left ventricular hypertrophy, as well as in combination with diabetes mellitus. A correlation analysis in patients with grade III obesity and AG revealed a close relationship between IGF-1 levels and left ventricular mass index. The results of the study showed that IGF-1 deficiency in women with hypertension and MS plays an important role in the development of myocardial remodeling and fibrosis, and prone to the development of heart failure. On the other hand, in obese women with hyperinsulinemia , IGF-1 production in the liver and myocardium is reduced. Against the background of its deficiency develops left ventricular hypertrophy. It is known that insulin is an important modulator of the effect of IGF-1, the effect of insulin on myocardial remodeling processes, apparently, both through its direct effect on IGF-1 receptors in cardiomyocytes, and indirectly by stimulating IFR-1 synthesis can be done. However, unlike insulin, which is not synthesized in the myocardium, local secretion of IGF-1 and its receptors occurs in cardiomyocytes due to its synthesis by autocrine or paracrine mechanisms . Therefore, myocardial remodeling, apoptosis, and the development of interstitial fibrosis in AG and obesity may occur with IGF - 1 mediation [22,25,28]. In addition, a physiological decline in growth hormone and

IGF-1 levels begins after puberty, which develops in proportion to a decrease in the hormonal function of the gonads [17]. Decreased levels of growth hormone secretion associated with aging may decrease IGF-1 synthesis and its levels in the blood and tissues. There is evidence that IGF-mediated involvement in the regulation of the normal menstrual cycle in young women, while IGF-1 expression in endometrial stromal cells is stimulated by estrogens. An increase in estradiol levels in the proliferative phase of the cycle leads to the stimulation of IGF-1 expression in the blood and endometrium, which then leads to its increase. It is known that estradiol levels decrease in postmenopausal women, so the proliferative effect of IGF-1 on the endometrium is reduced. IGF-1 levels in the blood and endometrium are significantly reduced. Therefore, against the background of hypoestrogenemia in postmenopausal women, IGF-1 deficiency leads to the development of obvious changes in the cardiovascular system, such as apoptosis and a decrease in the number of cardiomyocytes, remodeling of the myocardium and fibrosis. On the other hand, IGF-1 deficiency in patients with MS with MS, which develops against the background of severe obesity and hypoestrogenemia, significantly enhances the processes of myocardial remodeling and fibrosis, leading to the early development of heart failure. In addition, there is evidence that low levels of IGF-1 are a biochemical sign of deterioration of anabolic processes [40] and are a predictor of chronic heart failure decompensation, indicating an unfavorable prognosis and a high risk of death [31,33,35]. Thus, IGF-1 deficiency in postmenopausal patients with AG and MS is associated with a variety of structural, metabolic, and hormonal disorders, the most important of which is the development of hypertrophic forms of myocardial remodeling and subsequent heart failure.

AG and MS are related to the severity of obesity and reflect myocardial remodeling properties. In patients with hypertension and grade 1 obesity, concentric remodeling of the left ventricle predominated, and an increase in IGF-1 activity was noted.

The formation of hypertrophic types of myocardial remodeling at levels 2-3 of obesity is characterized by a decrease in IGF-1 levels, the minimum values of which were found in patients with AG with grade 3 obesity, with eccentric left ventricular hypertrophy predominating [22,30,31].

of IGF-1 on cell growth and differentiation processes, the participation of cytokine in the regulation of carbohydrate metabolism and the functioning of the cardiovascular system in obese patients is highly controversial. data are described. Thus, in a study by Zakirova NE et al (2017), high levels of IGF-1 were observed in patients with stage 1 AG and primary obesity, and a gradual decrease in blood pressure and TMI was observed. In another study, by contrast, the highest levels of IGF1 were observed in patients with combined AG and type 2 diabetes. However, IGF-1 levels showed a stable correlation with cytokine profile indicators, impaired carbohydrate metabolism, and diastolic dysfunction [13, 22,36].

Insulin-like growth factor 1 (IGF-1) plays an important role in the energy balance of the newborn’s body, it is associated with protein reserves, and its level is seen as an indicator of nutritional status. can exit. Based on the literature, it can be said that, in general, IGF-1 levels in premature infants are relatively low in premature infants. Analysis of the content of IGF-1 in the blood of premature infants showed that the values of this indicator in the neonatal period are very different: in the first study their range of values was 1.0-48.13 μg / l, in the second 3, 14–60, 3 mkg / l. Two variants of IGF-1 and blood nutrient dynamics of the studied nutrients were identified during early postpartum adaptation in preterm infants [39].

IGF-1 **is** a polypeptide hormone produced primarily by the liver in response to growth hormone stimulation. It is formed in smaller amounts in other organs and tissues and has an auto- or paracrine effect there. The nature of insulin, cortisol, and nutrition significantly affects hormone expression. Growth factor and IGF-1 functions are not fully separated.

IGF-1 acts on growth hormones, as well as has its own activity - anabolic, antioxidant, anti-inflammatory and cytoprotective effect. It affects the development, growth and differentiation of cells, tissue regeneration. Unlike the growth factor, IGF-1 levels are constant throughout the day and do not depend on food intake. Plasma IGF-1 concentrations are associated with moderate levels of growth

hormone throughout the day in both healthy individuals and patients with acromegaly . The amount of IGF-1 secreted depends on the basal level of growth hormone and is not related to the amplitude of its secretion peaks [24]. In infancy, IGF-1 promotes growth, neuro- and sympathogenesis, and overall development; in childhood it mainly affects bone metabolism, anabolic processes and proliferation; affects glucose and lipid metabolism in young and middle age, has antioxidant and anti-inflammatory properties, is a hepato- and cardioprotector; in old age it provides neuroprotection, mitochondrial protection, and reduces apoptosis. IGF-1 deficiency is observed in children with Laron syndrome (congenital short stature - due to a defect in the gene for somatotropic hormone receptors), cirrhosis of the liver in adults, cardiovascular and nervous system diseases in the elderly, and underdevelopment of fetal growth. The maximum concentration of IGF-1 is observed in the prepubertal period and early adolescence, over the years it gradually decreases. In healthy people over 65, IGF-1 secretion is 50-70% lower than in young people. The amount of IGF-1 depends not only on age but also on gender. In women aged 25-34 years, the concentration of IGF-1 is higher than in men, and vice versa at the age of 55-64 years. Blood IGF-1 concentrations are inversely related to age, body mass index, SAB, and total cholesterol levels in men and women. There is a positive correlation between IGF-1 composition and growth [7].

The components of the IGF-1 system, including itself, its receptors and binding proteins, are regulated by many factors, including other hormones, cytokines, lipoproteins, and hemodynamic load. In the uterus and ovaries, IGF-1 secretion is regulated by estrogens and follicle-stimulating hormone. Thrombin, tumor necrosis factor, and estrogens reduce IGF-1, mRNA production, and protein levels in smooth muscle cells.

Active forms of oxygen and angiotensin II have the opposite effect. The effect of LDL and platelet growth factor on IGF-1 secretion is unclear. IGF-1 promotes tissue growth, which is not possible without increasing their nutrition, which means increased blood flow. The effect of the hormone on blood vessels is associated with its vasodilating effect, as well as with the good absorption of glucose (including by microvascular endothelial cells), ie insulin-like effect. In vessels, IGF-1 is involved in the development of atherosclerosis, restenosis, diabetic injury, and angiogenesis [8]. IGF-1 is involved in the activation of vascular endothelial growth factor and stimulates endothelial cell growth. In experiments, the introduction of IGF-1 is accompanied by an increase in the density of cerebral vessels, and antibodies against it block this effect. IGF-1 is 50% similar to proinsulin and provides sensitivity to up to 10%. Both insulin and IGF-1 can activate each other’s receptors, but have a much lower affinity for foreign receptors than their own receptors. Decreased plasma IGF-1 concentrations are associated with the development of insulin resistance and metabolic syndrome, which are less common when IGF-1 and hydroxyvitamin D (calcifediol) concentrations are high . In metabolic syndrome, high levels of insulin lead to a decrease in IGF-1 production in the liver and tissues. A decrease in IGF-1 concentration, along with an increase in blood pressure, has a significant effect on the occurrence of vascular complications in diabetes. This is because IGF-1 may stimulate more smooth muscle cell migration and proliferation during hyperglycemia than euglycemia. IGF-1 contributes to the stabilization of atherosclerotic plaques by reducing oxidative stress, apoptosis, inflammatory signals, and endothelial dysfunction. Risk factors for the development of cardiovascular complications are increased PZXL, impaired insulin resistance, central obesity, smoking and AG, endothelial dysfunction with a decrease in IGF-1 levels [3].

are associated with an increased risk of ischemic heart disease, stroke, and heart failure [7]. Growth factor and IGF-1 deficiency worsen NO-dependent vasodilation associated with blood flow, increasing cardiovascular disease and mortality.

The side effects of IGF-1 disappear quickly after discontinuation of the drug. These include tachycardia, headache and vomiting as a result of a temporary increase in blood pressure, lipohypertrophy at the injection site, hypertrophy of the tonsils and adenoids, swelling of the face, arthralgia, myalgia, asthenia, orthostatic hypotension and hypoglycemia (due to activation of insulin receptors). enters. Although IGF-1 concentrations are often high in cancer, its use in low doses does not stimulate oncogenesis.

Patients with type 2 diabetes receive an increase in IGF expression when taking pioglite, which is an activator of PPAR- g receptors, which improves insulin sensitivity and reduces lipolysis in liver and adipose tissue, resulting in increased growth factor synthesis and, consequently, The formation of IGF-1 is also stimulated. IGF-1 has a significant effect on the condition of the cardiovascular system in adults.

And smooth muscle cells of the vascular wall , causes vasodilation, and has insulin-like properties. IGF-1 influences the development of diseases that play an important role in cardiovascular pathology - ischemic heart disease, hypertension and diabetes. Reduces the manifestation of insulin resistance in diabetes; may contribute to both plaque stabilization and growth in ischemic heart disease; causes vasodilatation, which lowers blood pressure in hypertension, and stimulates the growth of smooth muscle cells and cardiomyocytes, increases blood pressure.

Due to the wide range of multidirectional effects, the only recognized indicator of IGF-1 use in these diseases is clearly enhanced insulin resistance. In other cases, the combination of positive and negative effects requires a more in-depth study of the IGF-1 system to create drugs with minimal side effects [5].

PAPP-A is an acute phase protein that reflects atherosclerotic plaque damage. Increased concentrations of PAPP-A occur in acute coronary syndrome and massive vascular inflammation of atherogenic etiology. IGF-I is a growth protein that reflects the process of vascular repair. Increased IGF-I concentrations are associated with the development of ischemic heart disease and arterial hypertension [6,12].

The absence of gender characteristics of PAPP-A and IGF-I has been confirmed, and the patterns of gender-specific acute coronary syndrome outcomes have not been established. However, a negative statistical correlation was found between IGF-I levels and the age of patients with acute coronary syndrome . Thus, older patients have lower concentrations of IGF-I and a relatively low reparative ability to repair damaged vessel wall and myocardium.

Pregnancy-related plasma protein A and insulin-like growth factor 1 are new hypersensitive biochemical markers of vascular inflammation and injury. Their level can be used to predict atherosclerotic plaque instability in acute coronary pathology and disease prognosis [6].

Abbreviations:

IGF is an insulin-like factor

IGFBO is a protein that binds to an insulin-like factor

SBP - systolic blood pressure

DBP - diastolic blood pressure

References

1. Soliev A.U. / Prevalence of arterial hypertension and occurrence of risk factors in the elderly. // Journal of Biomedicine and Practice. B.435-441. Special issue. 2020. Tashkent.
2. Soliev A.U., Sobirov Sh. / Clinical Effects And Mechanisms Of Action Heparina - (Review Literature). Electronic scientific journal "Biology and integrative medicine" 2017 №4 (April). S.152-162
3. Soliev A.U., Rajabova G.X., Jumaev K.Sh. / Risk factors for arterial hypertension in elderly patients. // Asian Journal of Multidimensional Research. Volume 8, Issue 11, November 2019. P.75-80
4. Soliev A.U., Djumaev K.Sh., Radjabova G.X. / Treatment of bronchial asthma by physiotherapeutic methods. Theory and practice of modern science №5, 2018. C.783-787

5. Soliev A.U. Combination therapy of the bronchial hyperresponsiveness - Biology and integrative medicine, 2018. №2.S.57-66.
6. Soliev A.U. Combined therapy of hyperreactivity of the bronchi - Health status: medical, social and psychological-pedagogical aspects, 2016. p. 405-412
7. Brugts MP, Beld AW, Hofland lJ et al. Low circulating Insulin-like growth factor-1 bioactivity in elderly men is associated with increased mortality. J Clinical Endocr 2008; 93 (7): 2515-22.
8. Jun lA ., Scheike T., Davidsen M. et al. Low serum insulin-Like growth factor-I is associated with increased risk of ischemic heart disease: a population case-control study. Circulation 2002; 106 (8): 939-944.
9. Dronov VV, Sitnikov MY, Grinyova EN et al. Dynamics of growth hormone and IGF-1 in the blood of patients with decompensated congestive heart failure, a marker of prognosis and treatment efficacy. Serdechnaya Nedostatochnost '2013; 14 (6): 329-33
10. Le Roith D. The insulin-like growth factor system. Exp Diabesity Res. Oct-Dec 2003; 4 (4): 205-12.
11. Khodzhaeva D. I. Changes in the Vertebral Column and Thoracic Spinecells after Postponement of Mastoectomy //International Journal of Innovative Analyses and Emerging Technology. – 2021. – Т. 1. – №. 4. – С. 109-113.
12. Ilkhomovna K. D. Modern Look of Facial Skin Cancer //Барқарорлик ва Етакчи Тадқиқотлар онлайн илмий журнали. – 2021. – Т. 1. – №. 1. – С. 85-89.
13. Sultonova N. A. Treatment of hypercoagulable conditions in women with misscarriage in early gestation //Asian Journal of Multidimensional Research (AJMR). – 2020. – Т. 9. – №. 12. – С. 13-16.
14. Султонова Н. А. ИНДИВИДУАЛЬНЫЙ ПОДХОД К ПРОГНОЗИРОВАНИИЮ САМОПРОИЗВОЛЬНЫХ ВЫКИДЫШЕЙ У ЖЕНЩИН ДО 24 НЕДЕЛЬ ГЕСТАЦИИ //Современные вызовы для медицинского образования и их решения. – 2021. – Т. 406.
15. Султонова Н. А. ПРОГНОСТИЧЕСКИЕ КРИТЕРИИ САМОПРОИЗВОЛЬНЫХ ВЫКИДЫШЕЙ В СТРУКТУРЕ ПРЕВЫЧНОГО НЕВЫНАШИВАНИЯ НА РАННИХ СРОКАХ БЕРЕМЕННОСТИ НА ФОНЕ ПАНДЕМИИ COVID-19 //ЎЗБЕКИСТОН РЕСПУБЛИКАСИ СОҒЛИҚНИ САҚЛАШ ВАЗИРЛИГИ ТОШКЕНТ ТИББИЁТ АКАДЕМИЯСИ. – С. 60.
16. Uktamovna E. M. The Degree of Metabolic Disorders in Children and Adolescents with Obesity and Hypertension //Spanish Journal of Innovation and Integrity. – 2022. – Т. 6. – С. 136-141.
17. Uktamovna E. M. Diagnostic Markers of Cardiovascular Complications after Covid-19 //Spanish Journal of Innovation and Integrity. – 2022. – Т. 4. – С. 210-215.
18. Ibragimovna K. O. Competence of Quantitative Indicators of the Leading Clinical Signs of Cholestasis in Differentiation of its Grades //Indonesian Journal of Innovation Studies. – 2022. – Т. 18.
19. Kurbanovna S. I. THYMOGENIC IMMUNOCORRECTION OF CHILDREN WITH CONGENITAL HEART DEFECTS //ResearchJet Journal of Analysis and Inventions. – 2022. – Т. 3. – №. 1. – С. 34-43.
20. Kurbanovna S. I. Functioning of the Immune System in Children, After Surgical Correction of Congenital Heart Defects //European Journal of Life Safety and Stability (2660-9630). – 2021. – Т. 12. – С. 439-446.

21. Kurbanovna S. I. THYMOGENIC IMMUNOCORRECTION OF CHILDREN WITH CONGENITAL HEART DEFECTS //ResearchJet Journal of Analysis and Inventions. – 2022. – Т. 3. – №. 1. – С. 34-43.
22. Abdikaxhorovna K. F. Characteristics and Immunological Status of Chronic Jaundice in Infants Born to Mothers Who Passed Covid-19 //EUROPEAN JOURNAL OF MODERN MEDICINE AND PRACTICE. – 2022. – Т. 2. – №. 4. – С. 38-41.
23. Хамраева, Дилноза Раззаковна. "ЧАСТОТА РАСПРОСТРАНЕНИЯ ФУНКЦИОНАЛЬНЫХ ЗАПОРОВ У ДЕТЕЙ В БУХАРСКОЙ ОБЛАСТИ." Опыт и перспективы развития экспортного потенциала образовательных услуг в высшем образовании (2021): 227-232.
24. Karimovna, Turayeva Nargiza. "Predictive criteria for the severity of associated glomerulonephritis virus in children." CENTRAL ASIAN JOURNAL OF MEDICAL AND NATURAL SCIENCES 2.3 (2021): 295-299.
25. Харибова Е. А., Тешаев Ш. Ж. Морфофункциональные особенности тканевой организации энтероэндокринных клеток в возрастном аспекте //Проблемы биологии и медицины. – 2020. – №. 2. – С. 168-173.
26. Харибова Е. А. Особенности морфологии нейрональных ансамблей в тройничном узле человека //Морфология. – 2011. – Т. 140. – №. 5. – С. 123-124.
27. Махмудов З. А., Нечай В. В., Харибова Е. А. Железисто-лимфоидные взаимоотношения в стенке илеоцекального перехода на разных этапах постнатального онтогенеза //Морфология. – 2008. – Т. 133. – №. 2. – С. 85.
28. Файзиев Х. Б. Изменения морфологических параметров ткани селезёнки лабораторных крыс на 21 день после черепно-мозговой травмы средней тяжести //Central asian journal of medical and natural sciences. – 2021. – С. 109-113.
29. Файзиев Х. Б., Хусейнова Г. Х. Макроскопическая топография селезёнки белых беспородных крыс после тяжёлой черепно-мозговой травмы, вызванной опытом дорожно-транспортным происшествием //Проблемы биологии и медицины. – 2020. – №. 6. – С. 124.
30. Saidova L. B. et al. Optimization of medical care for patients with acute poisoning at the pre-hospital stage by emergency medical care team //Of XY international Research and practice conference England, London. – 2019. – С. 120-122.
31. Adizova D. R. et al. Rational approach to standard therapy //Central Asian Journal of Pediatrics. – 2019. – Т. 2. – №. 2. – С. 49-53.
32. Адизова Д. Р., Иброхимова Д. Б., Адизова С. Р. Приверженность лечению при хронической сердечной недостаточности //Биология и интегративная медицина. – 2020. – №. 6 (46). – С. 112-122